Chapter 13

Musculoskeletal Disorders

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I. DISEASES OF THE MUSCLES

A. Nutritional myopathy in young animals

1. Patient profile and history

a. Affected animals

(1) Nutritional myopathy is seen mainly in young, rapidly growing calves and lambs. This disorder occurs most commonly at 2–4 months of age but can occur in younger or older animals. It often accompanies unaccustomed exercise. Disease may occur in outbreaks wrth up to 15% morbidii and a 100% case fatality rate.

(2) In foals, it usually affects individual animals and is not associated with exercise.

(3) Myopathy may occur with vitamin E and selenium deficiency in piglets but is overshadowed by other major pathologic findings (e.g., mulberry heart disease, hepatosis dietetica).

b. The condition is most common in selenium-deficient areas (e.g., northeast and northwest North America) and in dams that are fed winter diets deficient in vitamin E and selenium. Occurrences may be associated with diets containrng unsaturated fats (e.g., fish oils, corn oils).

2 Clinical findings

a. Calves and lambs

(1) Acute **enzootic** muscular dystrophy. Calves and lambs may die suddenly. If the animals survive, they may be found in lateral recumbency. They are seen to be bright, alert, and usually able to swallow. Tachycardia (150–200 beats/minute) is a feature often coupled with an irregular heart beat

(2) Subacute **enzootic** muscular dystrophy. The most common presentation of nutritional myopathy is white muscle disease in calves and lambs.

(a) Animals present in sternal recumbency and are bright, alert, and unable to stand. If animals are standing, they are weak, stiff, and trembling.

(b) On bilateral palpation, muscles may be firm and swollen, particularly over the gluteal and shoulder muscles. Calves and lambs are often dyspneic because of intercostal muscle involvement, and a transient fever (41°C) may be present. There is tachycardia, but the heart rhythm is regular.

b. Foals. Myopathy may occur in foals from 1 week to 5 months of age. Clinical expression is recumbency, difficulty in rising, trembling, unsteadiness, failure to suckle, polypnea, and tachycardia. A specific localized presentation occurs where face and neck muscles degenerate. Therefore, foals may appear to have a stiff neck and be unable to nurse.

3. Etiology and pathogenesis

a. Etiology. Although their exact relationship is unknown, dietary selenium, sulfur-containing amino acids, and vitamin E work together to protect cells from oxidative damage. Therefore, a deficiency in these agents can cause nutritional myopathy.

b. Pathogenesis

(1) Selenium is an essential component of **glutathione peroxidase (GSH-Px).** GSH-Px detoxifies lipid peroxides by reducing them to nontoxic hydroxy fatty acids. A deficiency of selenium limits the production of GSH-Px and other selenoproteins, thereby allowing a buildup of oxygen (free radicals), which causes cellular destruction.

(2) Vitamin E prevents fatty-acid hydroperoxide formation and protects cellular membranes from lipoperoxidation. High levels of dietary unsaturated fatty acids may overwhelm the protective mechanism of vitamin E but are not a prerequisite for disease. Selenium may limit the amount of dietary vitamin E required.

(3) Affected muscles. Presentations may not be distinct and can occur together in the same animal.

(a) In young lambs, calves, and foals, myocardial and diaphragmatic muscles are most often affected, resulting in a presentation of acute heart failure, respiratory distress, and rapid death.

(b) Older animals, particularly calves, often present with skeletal muscle involvement.

4. Diagnostic plan

a. Acute cases. The diagnosis for acute enzootic muscular dystrophy is often based on postmortem findings, but laboratory results are diagnostic in the live animal.

b. Subacute cases. The diagnosis for subacute enzootic muscular dystrophy is based on clinical findings and dramatically increased muscle enzymes and decreased serum selenium and GSH-Px.

5. Laboratory tests

a. Plasma creatine phosphokinase (CPK) is the most commonly used laboratory measurement. CPK is highly specific for cardiac and skeletal muscle and will show a 10- to 50-fold increase in clinical cases of nutritional myopathy. CPK has a short half-life (2–4 hours) but will indicate muscle damage for up to 3 days.

b. Elevations of aspartate aminotransferase (AST) indicate muscle or liver damage. Levels remain high for 3–10 days at three to ten times the normal values. The magnitude of increase in both CPK and AST is proportional to the amount of muscle damage.

c. Serum and whole-blood selenium levels correspond to whole-body selenium status. Serum levels are the most reliable; however, neither test is available outside of research institutions or major reference laboratories.

d. Levels of the enzyme CSH-Px, which is found in red blood cells (RBCs), parallel the selenium status of the animal because selenium is incorporated into the erythrocyte during erythropoiesis.

e. Vitamin E status may be measured via a-tocopherol or vitamin E levels in the blood; however, these tests are less reliable as indicators of disease.

f. Myoglobinuria does not occur. Muscle mass is still limited at this age, and myoglobin content of muscle is low.

6. Differential diagnoses

a. Acute cases. Differential diagnoses include pneumonia, septicemia, and toxemia.

b. Subacute cases may appear similar to pneumonia, neurologic disease (e.g., meningitis), trauma, blackleg, or toxicities, as with organophosphates. In lambs, enzootic ataxia and swayback may appear similar.

7. Therapeutic plan

a. With the acute myocardial form, there is generally an unfavorable response to vitamin E and selenium therapy. Animals usually die within hours despite therapy.

b. For the subacute form, a single treatment of the recommended therapeutic dosage of a vitamin E and selenium preparation at 2 ml/45 kg is curative. Animals respond to therapy in 2–5 days.

8. Prevention. Dietary supplementation with vitamin E and selenium is necessary in deficient geographic locations. During a dam's pregnancy, this supplement may be provided in the feed as mineral or salt mixtures. Calves should be treated intramuscularly at birth (and 4–6 weeks later if necessary) with vitamin E and selenium at 1 ml/45 kg.

B. Myopathy in older animals

1. Muscular dystrophy (paralytic myoglobinuria) of cattle

a. Patient profile and history. This myopathy is seen sporadically in cattle up to 2 years of age. There is a recent history of increased exercise and a patient profile of heavily fed, well-muscled animals.

b. Clinical findings. Affected animals are found in lateral recumbency, with a bright and alert attitude. Skeletal muscles may feel firm, particularly the semimembranosus and semitendinosus muscles. Myoglobin may be present in the urine, which is brown.

c. Etiology and pathogenesis are the same as with young animals (see I A 3).

d. Diagnostic plan and laboratory tests

(1) The preliminary diagnosis is based on clinical findings and history. Other causes of recumbency (e.g., trauma, malnutrition) must be eliminated.

(2) The diagnosis is confirmed through clinical pathology findings. CPK is very high in clinical cases (usually more than 20,000 IU/L). Vitamin E levels are low in herdmates.

e. Therapeutic plan

(1) This condition is treated with parenteral solution of vitamin E and selenium at recommended dosages.

(2) It is important to maintain deep bedding while the animal is recumbent to limit pressure necrosis of muscles. Recumbency may be prolonged for days or weeks during convalescence, which clouds the prognosis and limits economic salvage of affected animals.

f. Prevention. Dietary supplementation with vitamin E and selenium is recommended for the herd.

2. Exertional (post-exercise) rhabdomyolysis in horses

a. Patient profile and history

(1) The most common presentation of the condition (also called equine paralytic myoglobinuria, tying-up syndrome, and azoturia) is in horses after unaccustomed exercise or insufficient training.

(2) There may be a familial basis to the disease in certain breeds (e.g., Quarter horse), but the condition is common among all breeds and is overrepresented in young, female animals.

(3) The condition may be chronic and recurring in certain animals.

b. Clinical findings. There is a sudden onset of muscle soreness, which may range from stiffness (a rigid stance) to recumbency, and the animal sweats profusely after exercise. There is swelling and rigidity of various skeletal muscle groups. Myoglobinuria is evident clinically.

c. Etiology and pathogenesis

(1) Etiology

(a) Predisposing factors include heavy musculature, irregular exercise, high-grain diet, and a nervous disposition. There is degeneration of muscle cells and muscle bundles associated with unaccustomed exercise (e.g., Monday morning disease).

(b) The condition is not associated with the vitamin E or selenium status of the animal.

(2) Pathogenesis. Recent studies have indicated that the pathogenesis may be a glycogen storage disorder with a possible autosomal-recessive pattern of inheritance. However, there may be multiple causes for the accumulation of glycogen and polysaccharide.

d. Diagnostic plan and differential diagnoses. Clinicians should rely on history, clinical findings, and laboratory results to establish the diagnosis. For chronic or relapsing cases, a muscle biopsy may be useful in differentiating the clinical picture from that of lameness.

e. Laboratory tests. There is an increased CPK level (5000–10,000 IU/L) and myoglobinuria.

f. Therapeutic plan

(1) Early therapy is essential, including rest, analgesics, and supportive therapy (e.g., bedding, intravenous fluids if necessary to increase excretion and dilute myoglobin).

(2) Sodium bicarbonate intravenously is warranted in cases with acidemia.

g. Prevention. The only recommended prevention includes training modifications and attention to diet. Overconditioned animals are more prone to exertional

rhabdomyolysis. Dantrolene sodium has been used to slow calcium release and decrease muscular contraction.

3. Ischemic myopathies
   a. Patient profile and history. Large animals in prolonged recumbency on hard surfaces (e.g., horses following anesthesia, downer cows with hypocalcemia or calving injuries) are most prone to this condition.
   b. Clinical findings. Hind limbs or areas of pressure during recumbency are affected. Animals appear bright and alert with normal appetite but are unable to rise. Cattle may creep along, pulling themselves forward using their front legs.
   c. Etiology. Ischemic myopathy is caused by muscle degeneration resulting from local ischemia, Prolonged muscle compression results in tissue anoxia, cellular damage, inflammation, cell death, and muscle degeneration.
   d. Diagnostic plan. The condition is diagnosed by a combination of history, clinical findings, and laboratory results. The clinician must rule out trauma or other causes of recumbency.
   e. **Laboratory** tests. Muscle enzymes are elevated but less so than with nutritional or exertional myopathies. Occasionally, post-anesthetic myopathies can cause great elevations in CPK, where the serum enzyme level may exceed 100,000 IU/L.
   f. Therapeutic plan
      (1) Consider the administration of analgesics, and institute supportive care (e.g., deep bedding, rolling, limited lifting or slinging).
      (2) Immersion in a specially designed water tank aids in physiotherapy for down cows. Horses have responded occasionally to the use of an equine swimming pool.
      (3) Correction of any fluid or electrolyte imbalance and administration of intravenous fluids to increase excretion of myoglobin are necessary therapies.
   g. Prognosis for individual cases is highly variable and must take into account the length of recumbency, muscle mass and size of the individual, flooring and surface characteristics, as well as attention to supportive care.
   h. Prevention. Advise the client regarding the primary cause (e.g., hypocalcemia), and institute appropriate preventive measures for the herd.

4. Porcine stress syndrome [PSS, pale soft exudative (PSE) pork, malignant **hyperthermia**]. It is thought that these terms are all **synonymous.** The names correspond to the stage of identification.
   a. Economic implications. This condition concerns the meat-packing industry because the keeping quality and presentation of cuts of pork are affected by the soft, pale, weeping condition of the meat [pale soft exudative (PSE) pork].
   b. Patient profile and history
      (1) If the condition is seen in finishing pigs, it is characterized by acute death induced by stressors (e.g., transport, fighting, high environmental temperature).
      (2) This disorder may also be seen in other species associated with the administration of anesthetics or muscle relaxants.
   c. Clinical findings
      (1) In pigs, the most dramatic sign is acute death, often after transport. If animals are found alive, they appear stiff or recumbent with tremors and have a high rectal temperature, dyspnea, and skin blanching with areas of erythema.
      (2) In other animals with an anesthesia-precipitated event, the signs are similar.
   d. Etiology and pathogenesis
      (1) Etiology. The condition is caused by an inherited **susceptibility** (single recessive gene) in all breeds of pigs but is most common in Landrace, Pietrain, and Poland China pigs and particularly in pigs selected for heavy muscling and rapid growth.
      (2) Pathogenesis. Biochemical events include a rapid onset of anaerobic glycolysis and loss of control of skeletal muscle metabolism. This results in excessive lactate production and the development of heat, which, in conjunction with peripheral vasoconstriction, results in hyperthermia.
   e. Diagnostic plan and **differential diagnoses.** Differential diagnoses are key to primary diagnosis. The clinical and postmortem findings help rule out other causes of sudden death, such as heatstroke.
   f. Laboratory tests. Clinical pathology is of little relevance in acute disease because of the rapidity of disease progression.
   g. Therapeutic plan. There is no treatment other than cooling and the immediate relief of stress; however, this therapy is invariably futile in clinical cases.
   h. Prevention
      (1) Screening
         (a) For many years in test stations, the halothane test was used to test for expression of the gene causing the condition but was only effective in revealing homozygous carriers. Test matings were then established to identify heterozygotes.
         (b) Other screening methods include blood creatine kinase levels, blood typing, meat quality evaluation for PSE pork, and erythrocyte fragility. The most recent test used to identify carriers is the ***DNA-based*** assay (gene probe) for a C–T mutation at base pair 1843 of the skeletal muscle ryanodine receptor (ryr1). This mutation is highly correlated with PSS.
      (2) Control of the condition in a susceptible population includes the reduction of stressors (e.g., ventilation, spray cooling, rapid transport). **Tranquillizers** have also been used, including azaperone (40–80 mg). Dantrolene sodium has been used prophylactically. It is hypothesized to slow the release of cellular calcium, thus limiting muscular contraction.

5. Other conditions that can cause myopathy in older animals include:
   a. Neurogenic myopathies (Akabane virus)
   b. **Neoplasia** (rhabdomyosarcomas)
   c. Congenital myopathies (double muscling)

C. **Myositis** of domestic animals. Myositis is manifested as either an acute inflammation with muscle swelling and pain or a chronic inflammation manifested by atrophy and joint and limb contracture. Many cases are traumatic in origin, with specific clinical signs and therapies related to the type and location of trauma.

1. **Clostridial** myositis (**clostridial** myonecrosis, blackleg, malignant edema)
   a. Patient profile and history. Clostridial myositis may follow intramuscular injections, which cause tissue necrosis. This has been described most frequently in horses.
      (1) Clostridial **myonecrosis** occurs primarily in cattle, sheep, goats, and wild ruminants. There is no sex or breed predilection. This is most often a disease of prosperous animals older than 3 months in age.
      (2) Blackleg
         (a) Cattle may develop resistance to ***Clostridium chauvoei*** after 2 years of age. The term blackleg most often refers to gangrenous myositis, which is caused by *C.* chauvoei.
         (b) In sheep, this infection is associated with wounds such as those associated with shearing, docking, and castrations, particularly in unsanitary conditions.
      (3) Malignant edema is more likely to be associated with wound infection (*C. septicum, C. novyi* type B, and occasionally *C. sordellii* and *C. chauvoei*). This disease usually affects sheep; however, it can affect other species of all ages.
   b. Clinical findings
      (1) Blackleg
         (a) Cattle. Usually, this disease is characterized by sudden death.
            (i) Clinical signs may include lameness, depression, anorexia, rumen stasis, reluctance to move, and fever (40°C).
            (ii) Muscle swelling resulting from subcutaneous emphysema can be seen or felt on the thigh, rump, loin, or brisket. Crepitation occurs on palpation. Swellings may be warm but progress rapidly to cold. The skin over these areas feels dry, is insensitive to pain, and is

discolored (blue, black). The course rapidly progresses to dyspnea, prostration, coma, and death.

(b) **Sheep.** The clinical signs are similar to those in cattle.

(2) **Malignant edema** presents as a rapidly spreading edema, and sometimes emphysema is seen around the site of a wound. Blood-tinged edema fluid gravitates from the wound to dependent areas through tissue planes. Death is rapid with this disease.

(3) ***C. sordellii*** infection produces a highly fatal myositis and can cause death in cattle and horses. The disease course is rapid, and clinical signs often do not develop before death.

c. **Etiology and pathogenesis**

(1) **Etiology.** Clostridial diseases are infectious but not contagious. *Clostridium* organisms are common inhabitants of the gastrointestinal tract but are mainly found as soilborne organisms. They are a constant threat to livestock, particularly those not in confinement.

(2) **Pathogenesis.** These organisms may persist in soil as spores for years and gain access to the animal through injections, wound contamination, or puncture wounds. If introduced as spores (particularly via the gastrointestinal tract), the organisms may sequester in tissue until a suitable anaerobic environment occurs (e.g., trauma, circulatory disturbances). Exotoxins elaborated by the organisms cause further necrosis, which encourages more bacterial growth.

d. **Diagnostic plan**

(1) The disease usually is diagnosed by **postmortem findings;** however, postmortem decomposition is particularly rapid with these diseases, and differentiation of organisms is difficult, particularly with overgrowth of other clostridia.

(2) Diagnosis is aided by an **accurate history** (e.g., occurrence of disease in the area, no vaccination history) and **tissue smears** from fresh carcasses.

e. **Therapeutic plan.** If clinically ill animals are observed, penicillin (44,000 IU/kg twice daily) or broad-spectrum antibiotics are administered for 5–7 days. The mortality rate with these diseases approaches 100%.

f. **Prevention.** In clinical cases in a herd, all exposed animals should be treated with a **multivalent clostridial vaccine.** Herd or flock immunization is effective and inexpensive. It should be practiced regularly, particularly in areas known to have outbreaks of clostridial diseases.

2. **Other conditions**
   a. **Eosinophilic myositis**
   b. **Fibrotic myopathy**
   c. **Ossifying myopathy**
   d. **Parasitic myositis**

II. DISEASES OF THE BONES AND JOINTS

A. Osteomyelitis

1. **Patient profile and history.** Osteomyelitis affects all species but is most common in young animals.

2. **Clinical findings.** Initial presentation includes a soft, sometimes painful lesion over bones or joints. The pain persists and becomes more severe (lameness), resulting in cellulitis, discharge from the sinuses, and pathologic fractures.

3. **Etiology.** The infection is introduced traumatically or hematogenously.
   a. **Specific causes** include spinal abscesses from tail docking, tail-biting pigs, and omphalophlebitis or septicemias that result in infectious arthritis, meningitis, and osteomyelitis.
   b. In **foals,** the cause is usually a hematogenous source for suppurative polyarthritis and concurrent osteomyelitis. There is physeal, metaphyseal, and epiphyseal involvement. Septicemia does not have to be clinically apparent. Causative organisms include *Escherichia coli, Actinobacillus equuli, Rhodococcus equi,* and others.
   c. **Horses.** In adults, open fractures commonly result in osteomyelitis.
   d. In **calves,** the cause may be similar to that in foals. Lesions are usually restricted to physeal or metaphyseal involvement.
   e. **Cattle.** A common osteomyelitis in cattle is **lumpy jaw** (see II B), which is caused by ***Actinomyces bovis.*** Traumatic causes are also common because of a variety of organisms, commonly ***A. pyogenes.***

4. **Diagnostic plan and laboratory tests.** Diagnosis is based on clinical findings, radiographic changes, cytology of an aspirate from the lesion, and possibly culture.
   a. **Bacterial culture.** In the case of concurrent arthritis, consider a **synovial biopsy** for bacterial culture. A synovial biopsy is often more likely to yield a positive culture than the fluid aspirate from a joint.
   b. A **blood culture** should also be performed in septicemic or bacteremic animals.

5. **Differential diagnoses** should include trauma, arthritis, and osteodystrophy.

6. **Therapeutic plan**
   a. **Medical therapy** may arrest clinical progression but is rarely curative. Long-term treatment is necessary with broad-spectrum antibiotics. Anaerobes may be involved, therefore, consider penicillin or metronidazole as an adjunct to other antibiotics (e.g., trimethoprim-sulfas). Intra-articular antibiotics have not proven to be more efficacious than systemic.
   b. **Surgery** is often the treatment of choice and involves arthrocentesis, surgical drainage, irrigation, and bone curettage or removal. Polysulfated glycosaminoglycan should be used as intra-articular therapy following surgical intervention.

7. **Prevention.** Proper neonatal management, attention to wounds, and early treatment are essential.

B. Actinomycosis (lumpy jaw)

1. **Patient profile and history.** Lumpy jaw is most common in cattle, although occasionally it is seen in pigs and horses. This disease is usually sporadic in young adult cattle and can occur in calves. There may be a farm or animal predisposition.

2. **Clinical findings**
   a. **Swelling.** Lumpy jaw presents as a **painless, bony swelling,** usually of the mandible and occasionally of the maxilla. The result is disfigurement that leads to improper mastication, which causes a loss of body condition.
   b. **Lesion.** The lesion is usually most prominent on the lateral aspect of the jaw but may occur on the inside of the mouth. Progression of the lesion is variable. In advanced cases, pus is discharged from various fistulae. The pus is described as "sticky" and "honey-like" and may be granular.
   c. **Soft tissue.** Involvement of soft tissue may occur, with clinical signs dependent on the area involved (e.g., lower esophagus).

3. **Etiology and pathogenesis**
   a. **Etiology.** The causative organism is ***Actinomyces bovis.***
   b. **Pathogenesis.** *A. bovis* is a common inhabitant of the bovine mouth, gaining entrance to subcutaneous tissues through compromised mucosa (trauma). Also, foreign bodies, plants, or infected teeth may aid in the introduction of infection. *A. bovis* also may invade the alveolus via erupting teeth.

4. **Diagnostic plan.** The diagnosis is based on clinical findings, smears of discharging pus, and granules submitted for microbiologic staining.

5. **Therapeutic plan.** All that can be hoped for is an arrest in the progression of the lesion. Many of the listed therapies can be carried out simultaneously in order to carry an animal for a short period of time (e.g., to calving).
   a. **Iodides** are recommended but are less effective with advanced lesions than early in the course of disease. Potassium iodide (6–10 g/day orally for 7–10 days) or

sodium iodide (1 g/12 kg intravenously as a single treatment, may repeat once) are used.

b. Sulfa drugs (1 g/kg daily for 4–5 days), streptomycin (5 g/day for 3 days intramuscularly), or isoniazid (10–20 mg/kg, orally or intramuscularly, for 3–4 weeks) may also be tried.

c. Surgical debridement also has been used.

6. Prevention. Cleanup of the environment is recommended to remove sharp foreign objects.

C. Osteodystrophy

1. Patient profile and history. Defective or abnormal formation of bone can occur in all species and is usually most evident in groups of animals (adults or young).

2. Clinical findings are usually less marked in adults than in young animals.
   a. Bone curvature and shifting lameness are the earliest clinical signs (bowing of long bones) and are most common in young, growing animals. Also, there is an enlargement of the distal ends of long bones. Epiphyses may be painful to palpation.
   b. Unexpected fractures may occur commonly in mature animals. The rib cage becomes flattened, and the animal appears "slab-sided."
   c. Other clinical findings occur with primary deficiencies or imbalances of certain nutrients (e.g., infertility, unthriftiness, teeth abnormalities).

3. Etiology and pathogenesis
   a. Etiology
      (1) Nutritional causes
         (a) Imbalances or deficiencies of calcium, phosphorus, and vitamin D, alone or in combination, produce bony abnormalities. Examples include rickets in young animals, osteomalacia in adult ruminants, and osteodystrophia in pigs and horses.
         (b) Other primary nutritional causes include copper deficiency (e.g., osteoporosis in lambs, epiphysitis in young cattle) and inadequate dietary protein, which leads to osteoporosis. Hypo- or hypervitaminosis A produces skeletal abnormalities in pigs and cattle.
         (c) Secondary protein or mineral imbalances. Chronic parasitism produces skeletal abnormalities resulting from protein deficiency. High levels of aluminum in the ration interfere with calcium and phosphorus metabolism.
      (2) Chemical agents
         (a) Chronic lead poisoning leads to osteoporosis in lambs and foals.
         (b) Chronic exposure to fluorine results in osteoporosis and exostosis.
      (3) Poisonous plants. Plants can interfere with mineral balance. For example, oxalate accumulators (e.g., rhubarb, sorrels, wild dock) can cause skeletal disease because of oxalic acid's great affinity for calcium and magnesium in the gastrointestinal tract.
      (4) Inherited or congenital conditions. Some known conditions include:
         (a) Achondroplasia and chondrodystrophy (dwarfism)
         (b) Osteogenesis imperfecta
         (c) Osteopetrosis
         (d) Arthrogryposis
         (e) Splayleg in piglets
         (f) Angular limb deformities
         (g) Flexural deformities
         (h) Ovine hereditary chondrodysplasia in Suffolk lambs (spider syndrome)
      (5) Environmental causes
         (a) Indoor housing with hard flooring has been associated with osteochondrosis, arthrosis, and epiphysitis.
         (b) Vertebral osteochondrosis or spondylitis is a described syndrome of older bulls in breeding units.
      (6) Unknown causes. Hypertrophic pulmonary osteoarthropathy is a disease of increased bone formation and has been reported in horses, cattle, and sheep. The cause, although unknown, is related to pulmonary lesions, such as tumors or chronic infectious processes.
   b. Pathogenesis
      (1) Imbalances of calcium, phosphorus, and vitamin D
         (a) Absorption. Calcium is absorbed from the small intestine according to need. Absorption is influenced by nature of diet, amounts of calcium and phosphorus in the diet, and requirements of the individual. Phosphorus is available in forage and grain diets.
         (b) Metabolism of calcium and phosphorus is under the control of parathyroid hormone and vitamin D. Parathyroid hormone is secreted in response to hypocalcemia and stimulates conversion of 25-dihydroxycholecalciferol to 1,25-dihydroxycholecalciferol (1,25-DHCC). Together, these stimulate bone resorption, and 1,25-DHCC alone stimulates intestinal absorption of calcium. This activity is balanced by calcitonin when serum calcium levels return to normal.
      (2) Copper deficiency causes changes in osteoblastic activity and impairment of collagen formation.
      (3) Fluorine toxicosis. Chronic ingestion of fluorine causes deposition in bones and teeth. Calcium and phosphorus are excreted in the urine in conjunction with fluorine, resulting in osteomalacia, osteoporosis, and exostosis.

4. Diagnostic plan. It may be difficult to define a specific disease entity, deficiency, or imbalance unless there are obvious characteristics of a specific condition (e.g., big head in horses, rickets in sheep). Conditions usually are diagnosed on the basis of history, clinical findings, laboratory results, feed analysis, and necropsy.

5. Laboratory tests. Serum calcium and phosphorus are not reliable indicators of primary disturbance or whole-body status, Combinations of feed samples, pathology specimens, blood chemistry, and urine chemistries (fractional excretions of electrolytes) may add to diagnostic evidence. For example, serum copper levels may be combined with a liver biopsy to determine copper reserves in suspected cases of epiphysitis due to copper deficiency.

6. Therapeutic plan
   a. Early treatment of animals affected by a dietary imbalance may yield positive results if lesions are not too far advanced or if the primary disease is treatable. For example, nutritional secondary hyperparathyroidism carries a better prognosis than renal secondary hyperparathyroidism.
   b. Specific conditions may respond to medical therapy. For example, selected cases of fetlock flexural limb deformities in foals often respond to intravenous tetracycline injection combined with splinting, foot trimming, phenylbutazone, and a slow return to exercise. The recommended dosage of intravenous tetracycline is a single 3-gram dose that may be repeated in 24 hours if there has been no response to therapy.
   c. Treatment of the group requires nutritional counseling and dietary management (supplementation or balancing). A balanced diet with adequate protein and good-quality roughage is extremely important in the growing animal. Injectable calcium, phosphorus, and vitamins may be considered.

7. Prevention is based on the specific diagnosis but often centers on nutritional management.

III. LAMENESS

A. Lameness of cattle

1. Foot rot (pasture foot rot, infectious pododermatitis, interdigital phlegmon, interdigital necrobacillosis). The disease is sporadic, but up to 25% of cows may be affected per year, making this an important economic disease.

a. Patient profile and history
   (1) This is a contagious and relatively common condition that affects mainly mature dairy cattle that are on pasture or living in unsanitary housing conditions.
   (2) This disease is seen most commonly during wet periods and under management conditions that cause abrasions between the claws (e.g., stones, rough ground, sharp objects).
b. Clinical findings
   (1) There is the sudden appearance of obvious foot lameness almost exclusive to the hind limb. The cow's temperature ranges from 39°C to 40°C, with a loss of milk production, loss of condition, or both. The limb bears little weight.
   (2) Heat, pain, and swelling of the coronet and between the claws is obvious. On closer examination, the lesion is seen to be a fissure in the interdigital space. The fissure is moist, red, swollen, and has a characteristic foul odor. If left untreated, this fissure may progress to produce deeper involvement of soft tissues, bones, and joints. In these cases, there is more severe lameness with swelling higher in the leg.
c. Etiology. It is thought that injury or compromise to the skin of the interdigital space (e.g., constant moisture) predisposes the animal to infection by ***Fusobacterium necrophorum.*** Other bacteria may be involved or may occasionally be isolated (e.g., *Bacteroides melaninogenicus,* Dichelobacter *nodosus*), but these organisms are not a requirement for disease.
d. Diagnostic plan. The diagnosis is based on history and clinical findings of a moist, red fissure in the interdigital space.
e. Differential diagnoses that are usually ruled out by clinical examination include traumatic lesions, hoof growth abnormalities, laminitis, and stable foot rot.
f. Therapeutic plan. Parenteral antibiotics and local treatment of early lesions often achieve success.
   (1) Procaine penicillin G (22,000 IU/kg intramuscularly, twice daily for 3–5 days) is often the treatment of choice. Other broad-spectrum antibiotics may be used but are no more successful (remember milk withdrawal). Occasionally, a highly resistant strain of *F.* necrophorum may be encountered, which limits the success of therapy.
      (a) Cows should be kept inside with dry footing until systemic therapy is complete.
      (b) Non-milking cattle may respond to long-acting antibiotics to reduce the number of parenteral injections necessary.
   (2) Local treatment. The foot should be examined by rope lift, chute restraint, and chemical tranquilization. Disinfectant creams or solutions (e.g., copper sulfate-5% paste) may be applied following thorough cleaning and debridement. Bandaging usually is not necessary in uncomplicated cases.
   (3) Surgery. For deeper involvement of tissues (synovitis, tenosynovitis, arthritis, osteomyelitis), surgical therapy may be necessary.
g. Prevention
   (1) Employ pasture modifications if possible (e.g., rotate pastures more often, fill mud holes, remove sharp objects).
   (2) With herd involvement, provide a foot bath of a 5%–10% solution of formaldehyde and copper sulfate in an area where cattle must walk (e.g., doorway). Copper sulfate may be used alone. Animals may try to drink the solution, so restrict access. Alternatively, dry lime may be used in a walkway box to coat feet and decrease moisture and bacterial proliferation.
   (3) With feedlot cattle, the feeding of chlortetracycline or ethylene-diamine dihydriodide (EDDI) has been advocated with some empirical efficacy. However, with dairy animals, both antibiotic and iodide residues are a concern in milk.
   (4) Commercial vaccines are available but efficacy is yet unproven.

2. Stable foot rot (underrun sole)
   a. Patient profile and history. This condition occurs in cattle that are housed for long periods (i.e., stanchioned cattle). It is associated with poor hygiene, and as in pasture foot rot, hind claws are more commonly affected.
   b. Clinical findings. The animal may be lame and tends to stand with the hind feet placed further behind than usual. Inspection of the plantar surface of the foot reveals necrosis and erosion of the heel and horn at the heel, as well as lateral hoof wall overgrowth over the sole.
   c. Etiology and pathogenesis
      (1) Etiology. Conformationally weak pasterns, overgrown hooves, or lesions caused by chronic laminitis may shift weight bearing to the heel, thus predisposing the animal to the condition. The lesion begins as heel necrosis from which ***Fusobacterium necrophorum*** can be isolated. ***Dichelobacter nodosus*** can also be recovered in some cases.
      (2) Pathogenesis. The heel necrosis and the change in hoof angle and foot placement causes the hoof to overgrow, which in turn traps debris and necrotic material between the hoof wall and sole. The separated sole, impacted debris, and necrosis of the sole cause lameness in advanced cases.
   d. Diagnostic plan. Clinical examination is sufficient for diagnosis.
   e. Therapeutic plan. Remove all abnormal horn via trimming, and restore normal conformation to the foot.
   f. Prevention. Regular foot trimming and nutritional counseling regarding laminitis are two of the primary preventive practices that may limit the incidence of disease. Foot baths and attention to dry-lot hygiene may help control the secondary invaders.

3. Sole ulcer (pododermatitis circumscripta)
   a. Patient profile. This condition most commonly is seen in stabled cattle, with hard flooring and diet being likely risk factors.
   b. Clinical findings
      (1) The foot lesion is a circumscribed ulcer on the sole, exposing corium. It is usually of the hind limb on the lateral claw at the heel–sole junction. If an ulcer is not obvious on initial examination, it may be exposed by trimming.
      (2) The sole overlying the lesion feels spongy. Granulation tissue may be seen extruding through the sole defect, and severe lameness is often present.
   c. Etiology and pathogenesis. The pathogenesis is still somewhat debatable, and although there may be an anatomical or mechanical predisposition of the lateral claw, a localized ischemia resulting from chronic laminitis may be the underlying cause of the condition.
   d. Diagnostic plan. The diagnosis is made by examination with careful attention to both hind feet.
   e. Therapeutic plan
      (1) Remove undermined sole with trimming, and pare away granulation tissue.
      (2) Medications such as an astringent powder may be considered in combination with a pressure bandage to control granulation tissue.
      (3) The unaffected claw (medial claw) may be blocked up to provide relief to the affected claw while the animal walks or stands. Commercial blocks or slippers are available for this purpose.

4. Digital dermatitis (hairy heel warts, strawberry foot rot)
   a. Patient profile and history. This worldwide disease was first described in North America in the early 1980s. It is now common with a high herd prevalence, particularly in dairy herds. Milking cattle are most commonly affected.
   b. Clinical findings
      (1) The early lesion of the foot is an interdigital roughness and reddening. This progresses to a red, papilloma-like lesion with erect hairs. The lesion usually is confined to the hind feet at the point of the cleft of the heels or up to the dewclaws. Further progression results in continued proliferation with a slimy or velvety appearance and the isolation of secondary bacterial organisms.
      (2) Lameness is not a constant feature, and it is assumed that any pain associated with the disease is sporadic.
   c. Etiology. A spirochete-like bacteria is associated with this disease, although its

etiology is unproven. Reproduction of the condition has been unsuccessful. Because topical antibacterial solutions and antibiotics have proven somewhat successful in treating the condition, the bacterial cause has remained the most popular.

d. **Diagnostic** plan. Diagnosis relies on the clinical findings and is based on observation of the lesion.

e. Therapeutic plan. Because lameness is not always associated with the lesion, treatment of only lame cows is the most rational economic decision. Various topical therapies are used, including sprays containing tetracycline and lincomycin. Topical solutions may be paired with bandaging for best results.

f. Prevention. In herds where the disease is prevalent, formalin, tetracycline, or lincomycin footbaths or sprays may be used as a preventive. The disease is thought to be transmissible, so practices that limit the introduction of infected cattle into uninfected herds are recommended.

5. Laminitis (founder, diffuse aseptic **pododermatitis**) is a diffuse, aseptic inflammation of the corium.

a. Patient profile and history. Laminitis is a condition usually affecting young dairy cattle or feedlot cattle. This disease is seen occasionally in older dairy animals 2–3 months after parturition or associated with **periparturient** diseases (e.g., mastitis, metritis).

b. Clinical findings. This disease is recognized in several forms. Subclinical laminitis is perhaps the most common form, but in the clinical sense, it is recognized as either acute or chronic.

(1) Acute laminitis is seen sporadically in individual animals. Clinical signs of foot disease may be overshadowed by associated diseases.

(a) There may be severe lameness, stiffness, and pain in any or all legs and a reluctance to walk. The cow may stand with an arched back, forelegs extended, and hind legs underneath. Recumbency is common.

(b) There is swelling and tenderness of the heel bulbs and the coronary band. The sole may become soft, and trimming reveals subsolar hemorrhage and bruising.

(2) Chronic laminitis is more prevalent than recorded. Signs are mild or unobserved (subclinical). Clinical signs occur mainly in the hind feet and consist of wide, flat, long, and misshapen feet. The pasterns and heels tend to drop. Trimming reveals hemorrhage at the white line, beneath the cranial sole, or both.

c. Etiology and pathogenesis. The cause and pathogenesis is uncertain for any of the forms (acute or chronic, clinical or subclinical).

(1) Acute laminitis is associated with an acute systemic disease or carbohydrate overload (ruminal acidosis). Hemodynamic and vascular changes occur, which decrease the blood supply to the laminae likely by the establishment of arteriovenous shunts. Laminar degeneration occurs, and the sensitive laminae of the third phalanx (P_3) separates from the interdigitating laminae of the hoof. Clinical signs and destructive lesions are not as severe as in horses, but pedal rotation may still occur.

(2) Chronic laminitis is likely associated with heavy, interrupted feeding of diets high in grain. Chronic constrictive vascular lesions at the level of the laminae may cause blood **stasis** in the capillaries. This blood **stasis** results in stagnant hypoxia, which interferes with the production of keratohyalin and hoof growth.

d. Diagnostic plan. The diagnosis is made based on history, clinical findings, and foot trimming to identify subsolar hemorrhages and to rule out other causes of foot lameness.

e. Therapeutic plan

(1) For individual cases, treat associated diseases, and consider the use of nonsteroidal anti-inflammatory drugs (NSAIDs) if cleared for use in the species of animal under care.

(2) For chronic cases, the only treatment is trimming the affected feet, which is not usually warranted or practical.

f. Prevention

(1) Affected cattle become more susceptible to chronic misshapen feet, thus requiring frequent trimming. Laminitis may predispose to other conditions (e.g., sole ulcer).

(2) For chronic laminitis and herd or feedlot problems, the owner may have to reduce feeding. which affects herd production. A better alternative is to establish bunk management practices, which allow for more frequent feeding of smaller amounts of carbohydrates.

6. Other conditions

a. **Interdigital** dermatitis
b. Verrucose dermatitis
c. Trauma
d. Horizontal cracks
e. Sand cracks

B. Lameness of horses

1. Thrush is a moist exudative dermatitis of the central and lateral sulci of the frog.

a. Patient profile and history. Thrush is most common in adult horses that are kept in confinement under unhygienic conditions.

b. Clinical findings. Soft, spongy, disintegrated frog horn is observed in advanced cases. This finding is accompanied by a characteristic fetid odor. Clinical cases are characterized by lameness and, if advanced, inflammation of the coronet and discharge of pus from fissures in the coronet and heels may be evident.

c. Etiology and pathogenesis

(1) ***Fusobacterium necrophorum*** is commonly isolated from foot lesions, but the pathogenesis is debatable.

(2) Unhygienic conditions, lack of exercise, and poor foot care lead to accumulations of soil and fecal material in the hoof sulci.

(3) Lameness may predispose to the condition by decreasing weight bearing and natural cleaning of the sole or by causing improper hoof growth. Infection is confined to superficial layers but will affect deeper tissue in untreated cases.

d. Diagnostic plan. Diagnosis is based on clinical findings and the examination of the sole.

e. Therapeutic plan

(1) Loose and necrotic material must be pared away.

(2) Antiseptic products (e.g., iodines, copper sulfate) should be administered daily.

(3) Bandaging may be necessary.

f. Prevention. Educate the client regarding proper hygiene, foot care, picking out the feet, and trimming. Correct any chronic lameness.

2. **Laminitis** is a local, inflammatory condition of the foot that is often a manifestation of a systemic disturbance.

a. Patient profile and history. Equine laminitis occurs most commonly in ponies but can affect all horses and is more common in the spring. Overweight ponies on pasture, mares with retained placenta, and horses with acute systemic diseases are most susceptible to this disease.

b. Clinical findings

(1) Acute laminitis. Clinical signs may involve all feet, although the front feet are more commonly affected.

(a) Lameness may develop rapidly. The horse shifts its weight onto the hind limbs (under its body), while front limbs are placed out in front. When forced to walk, the horse walks with short strides and places the feet down quickly. Recumbency is common.

(b) Affected hoof walls feel warm, and a bounding digital pulse is felt. Pain is most prominent over the sole, as evidenced by hoof testers.

(2) Chronic laminitis. The chronic form may be seen in individual limbs, particularly in overweight horses.

(a) Clinical signs include recurrent bouts of variable lameness or of chronicity following the acute form.
(b) Classic hoof wall "rings" are seen, and a dropped or flattened sole with a long toe develops.

(3) Clinical description. Lameness caused by laminitis has been graded according to the following scale:
(a) Obel grade 1
(i) Foot discomfort without pain
(ii) A short and stilted gait at the trot
(iii) No lameness
(b) Obel grade 2
(i) Some discomfort and lameness as evidenced by a stilted gait at the walk
(ii) Forefeet lifted without difficulty
(c) Obel grade 3
(i) Reluctance to move
(ii) Resistance to lifting forefeet
(d) Obel grade 4. The horse does not move without being forced.

(4) Other clinical findings associated with laminitis include a heel-to-toe placement while walking and recumbency. There may be additional clinical signs found with the associated systemic disease.

c. Etiology and pathogenesis
(1) Etiology
(a) Diseases that produce systemic disturbances often result in laminitis. These diseases include endometritis, salmonellosis, and colitis X.
(b) Carbohydrate overload is also a common cause of the condition.
(c) Predisposing factors include:
(i) Excessive trauma resulting from concussive forces on hard surfaces (e.g., road founder)
(ii) Excessive weight bearing forced on a single limb after injury to the contralateral limb

(2) Pathogenesis
(a) The condition may be initiated by events such as grain engorgement, toxic metritis, and septic shock, which may allow circulating endotoxins to initiate the peripheral vascular response. This vascular response (e.g., changes in vascular resistance) deprives the laminar corium of blood supply, resulting in separation of the sensitive laminae at the junction of P_3 and the hoof. In addition to vasoconstriction, there may be platelet aggregation, microthrombosis, perivascular edema, and arteriovenous shunting.
(b) The effect is most clinically apparent at the distal aspect of P_3, which angles downward resulting in the characteristic radiographic evidence of pedal rotation seen with the disease. Also, disseminated intravascular coagulation (DIC) develops just before the lameness due to laminitis occurs, and corticosteroids are capable of inducing laminitis. These findings and the complexity of the disease discourages an overall accepted hypothesis from emerging.

d. Diagnostic plan and laboratory tests
(1) The diagnosis is often made based on the clinical signs and history.
(2) Radiology confirms changes to the angulation of P_3 and shows evidence of gross laminar degeneration. Radiographs should be taken early in the course of the condition and are used to monitor changes in P_3.

e. Therapeutic plan
(1) Acute form. Laminitis is an emergency. Treat any initiating disease (e.g., mineral oil for carbohydrate overeating, antibiotics for metritis). If response to therapy is not evident within 48 hours, a poor prognosis is warranted and therapy should intensify. With no response in 3–4 days, pedal bone rotation will have almost invariably occurred. Penetration of the sole may then follow, with resultant pedal osteitis or abscessation requiring heroic measures or euthanasia.

(a) Agents used for pain relief include:
(i) Phenylbutazone 8.8 mg/kg (4.4 mg/kg in ponies and foals), which should be given immediately and continue for 4–5 days in decreasing doses
(ii) Flunixin meglumine (1.1 mg/kg), which may be given as an anti-inflammatory or .25 mg/kg for its postulated anti-endotoxic activity
(iii) Aspirin (10 mg/kg daily)
(b) Treatments used to restore digital blood flow include:
(i) Acepromazine, which should be administered for its analgesic and a-adrenergic effects (40 mg every 4–6 hours)
(ii) Phenoxybenzamine as an a-adrenergic antagonist (2 mg/kg every 12 hours)
(iii) Isoxsuprine hydrochloride, which is used at a dose of .6 mg/kg twice daily
(iv) Dimethylsulfoxide (DMSO) for its effects as a free radical scavenger, an anti-inflammatory, analgesic, vasodilator, and inhibitor of platelet aggregation
(v) Sodium heparin at 44–66 IU/kg intravenously every 4 hours to prevent microvascular thrombosis
(vi) Nitroglycerine (2% paste); 15–30 mg applied over each digital vessel daily, for a total dose of 30–60 mg. Alternatively, nitroglycerine patches may be used at the rate of 1 patch every 24 hours.
(c) Nerve blocks (posterior digital nerve desensitization) may be used to localize the pain to the feet.
(d) Trimming the foot and padding and supporting the sole can be effective and helpful. However, removing shoes during the acute episode may be painful. The use of local nerve blocks may help decrease the pain and resultant release of more vasoactive substances. Lowering the heels will help bring a rotated P_3 in better alignment with the ground but will also increase the tension on the deep digital flex or tendon. Therefore, this is a controversial practice.
(e) Walking the horse is controversial. Walking may promote blood flow but may further traumatize the sensitive laminae.
(f) Dietary modification. In the case of carbohydrate engorgement causing laminitis, feed only grass hay until clinical signs disappear.

(2) Chronic form. Chronic laminitis is expensive to treat in terms of labor and cost.
(a) D-l methionine has been advocated to restore disulfide bond substrate for maintenance of the hoof wall pedal bone bond. Attention to trimming and shoeing is necessary to provide support and allow for optimal hoof growth and foot conformation.
(b) Hoof-wall resection may be necessary to return more appropriate conformation and angle to the hoof wall.

f. Prognosis
(1) Acute form. P_3 rotation has been used as a prognostic indicator for return to function. If the rotation is greater than or equal to 11.5 degrees, there is an unfavorable prognosis. If the rotation is 6.8–11.5 degrees, the prognosis is guarded. Less than this, there is favorable prognosis for return to function.
(2) Chronic form. The long-term prognosis for return to function is very poor.

g. Prevention
(1) Acute form. To prevent the disease, discuss predisposing causes and dietary management. Make clients aware of the recurrent nature of the condition in some instances. Horses that recover should not be put back into work for a minimum of 45 days.
(2) Chronic form. To prevent the recurrence of chronic laminitis, maintain good dietary management, reduce weight in overweight horses, and consider the use of support pads.

C. Infectious foot rot in sheep

1. Patient profile and history
a. Infectious foot rot is a significant, production-limiting disease of sheep, but it may

also occur in goats. There is a species variability in susceptibility and a variation in incidence associated with climate and environment.

b. The disease occurs most commonly on pasture during wet springs or at times of persistent moisture. Hot, dry conditions are unfavorable for the spread of disease.

c. Foot rot is more common in areas with heavy population pressures. It may occur as outbreaks with up to 75% morbidity. The disease is of economic importance because of the cost of treatment and loss of production.

2. Clinical findings

a. Early clinical signs include inflammation (moistness and swelling) of the interdigital space, a break at the skin–horn junction, and separation of the soft horn. Lameness is mild at this stage. The clinical condition progresses to severe lameness (non-weight bearing) with a foul odor. The horn becomes loose and sloughs, creating an underrun sole.

b. Virulent foot rot may exhibit systemic signs of fever and anorexia because the lesion extends to deeper tissue (osteomyelitis) with sloughing of the foot.

c. Intermediate foot rot has only moderate clinical signs.

d. Benign foot rot. Because there are various strains and serotypes of the causative organism (Dichelobacternodosus), infection may occur with mild or no clinical signs. This form is called benign foot rot or footscald.

3. Etiology and pathogenesis

a. Etiology

(1) ***Dichelobacter*** nodosus, the causative organism, has varying virulence (proteolytic activity) and produces a variety of clinical signs (virulent, intermediate, benign).

(2) Predisposing factors

(a) ***Fusobacterium necrophorum*** causes an initial interdigital dermatitis, which may resolve as the pasture dries up. However, this infection may predispose for invasion by D. nodosus.

(b) Trauma (e.g., stones, strongyloides parasites) may be a predisposing factor to invasion by *D.* nodosus.

(c) During times of moisture and warmth, the bacteria more readily penetrate the skin of the interdigital space.

b. Pathogenesis

(1) Route of infection. D. *nodosus* invades horn by producing keratolytic enzymes. Discharges from affected feet serve as a source of infection for other sheep. The condition spreads rapidly.

(2) Carriers. D. *nodosus* does not persist on pasture but may do so for years in the feet of carrier sheep. Carrier sheep usually are not lame but may have misshapen feet and a pocket of infection beneath the underrun sole. Consequently, the disease may be introduced into a flock by the purchase of an infected animal.

4. Diagnostic plan and laboratory tests. It is important to diagnose the condition accurately because of the expensive and dedicated measures necessary to treat and eliminate the condition.

a. The preliminary diagnosis is based on clinical findings.

b. Bacterial cultures confirm the diagnosis but must agree with clinical and epidemiologic findings.

5. Differential diagnoses. Conditions that should be ruled out include foot abscesses, foreign bodies, granulomas, fibromas, trauma, contagious ecthyma, bluetongue, ulcerative dermatosis, strawberry foot rot (dermatophilosis), and laminitis.

6. Therapeutic plan. All forms of the disease respond to therapy, but the condition may be extremely difficult to eradicate. Treatment includes:

a. Trimming to expose necrotic sole

b. Applying local disinfectant (e.g., 5% formalin, *10%* $ZnSO_4$, 5% $CuSO_4$)

c. Administering antibiotics (D. nodosus is susceptible to many antibiotics, and one treatment with penicillin is often all that is necessary if dry weather intervenes.)

d. Rechecking of feet if wet environment continues

7. Prevention. Eradication and control is the long-term goal when the disease has been diagnosed, but this is often difficult to achieve.

a. Eradication. The objective is to cull or cure infected animals and return them to clean pastures. When sheep have been removed from contaminated pastures, these pastures can be considered free of D. nodosus after 14 days. The disease is best eradicated during the dry season.

b. Vaccination is proving promising.

D Caprine arthritis-encephalitis (CAE)

1. Patient profile and history. This disorder is common in adult dairy goats and is prevalent in North America, Europe, and Australia. It is common to have a high prevalence of serological conversion within a herd or population with a much lower expression of clinical signs.

2. Clinical findings

a. Enlarged carpal joints (called big knee) are seen in goats older than 6 months of age. The onset of this arthritis may be acute or insidious, but lameness usually is not severe.

b. There is gradual weight loss with the development of a poor hair coat and swollen joints. Terminally, there is emaciation and recumbency.

c. Other clinical findings may include interstitial pneumonia or mastitis and development of a hard udder. The CAE virus also causes a leukoencephalitis in kids.

3. Etiology and pathogenesis

a. Etiology. The etiologic agent is a retrovirus (subfamily lentivirus), which has similarities to the virus of Maedi-visna.

b. Pathogenesis

(1) This disease is transmitted to kids principally through milk and colostrum, although cross-infection can occur. Between 65% and *81%* of the goats in most goat herds are seropositive to the CAE virus (CAEV).

(2) CAE is a **multisystemic** disease involving synovial-like connective tissue. It is a lymphoproliferative disease caused by continual viral stimulation and the resultant immune response. This disease produces a hyperplastic synovitis.

4. Diagnostic plan. The diagnosis is based on history, clinical findings, and laboratory tests.

5. Laboratory tests

a. Synovial aspiration yields a brownish red synovial fluid.

b. Cytologic evaluation reveals a mononuclear reaction with high cell counts.

c. An agar gel **immunodiffusion (AGID)** test and enzyme-linked **immunosorbent** assay **(ELISA)** on serum are positive.

d. Radiologic evidence suggests soft tissue changes, which progress to proliferative bony changes.

6. Differential diagnoses. Clinicians should rule out other infectious causes for arthritis (e.g., Mycoplasma, Chlamydia, *Corynebacteria*).

7. Therapeutic plan. There is no known treatment.

8. Prevention

a. Total eradication of CAEV from a goat herd is unlikely because vertical transmission does not seem to be the sole mode of spread and seroconversion may be delayed after exposure to the virus.

b. To decrease infection pressures in the herd, identify infected animals through serology, and cull or house separately. Separate kids at birth and feed either colostrum from negative dams or pasteurized colostrum/milk. Because antibodies from positive animals are not protective, there is no need to feed colostrum from seropositive animals. Continue regular serological survey of the herd.

STUDY QUESTIONS

Directions: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the **ONE** numbered answer or completion that is BEST in each case.

1. Which statement regarding nutritional myopathies is true? They:

(1) do not occur in foals or mature ruminants.
(2) are subclinical in nature only and produce losses due to poor performance.
(3) may be caused by deficiencies of vitamin A.
(4) are most common in animals found in the arid interior of North America.
(5) produce high elevation in serum creatine phosphokinase (CPK) levels.

2. Mature dairy cattle recumbent for prolonged periods of time on a hard surface develop which one of the following disorders?

(1) Ischemic myopathy
(2) Milk fever
(3) Obturator paralysis
(4) Fat cow syndrome
(5) Blackleg

3. Which of the following terms are used interchangeably in swine?

(1) Blackleg, malignant edema, malignant hyperthermia
(2) Malignant hyperthermia, porcine stress syndrome, pale soft exudative pork
(3) Pale soft exudative pork, blackleg, malignant edema
(4) Porcine stress syndrome, malignant hyperthermia, malignant edema
(5) Blackleg, pale soft exudative pork, porcine stress syndrome

4. The best test to establish the carrier state of porcine stress syndrome is:

(1) test mating of suspect boars to suspect sows.
(2) serum ionized calcium concentrations.
(3) a gene probe for a C–T mutation.
(4) analysis of muscle biopsy for fast-twitch fibers.
(5) an adrenocorticotropic hormone (ACTH) stimulation test.

5. Which one of the following descriptions characterizes blackleg in cattle?

(1) Sudden death
(2) Chronic lameness
(3) The presence of a contaminated wound
(4) Overrepresentation in housed dairy cattle
(5) Resistance to herd level immunization

6. In young foals, the most common cause of osteomyelitis is:

(1) trauma inflicted by the mare.
(2) improperly placed vitamin injections.
(3) consuming mastitic milk from the mare.
(4) trauma during the foaling process.
(5) hematogenous spread of infectious organisms.

7. The organism most commonly associated with causing pasture foot rot in cattle is:

(1) *Dichelobacter nodosus.*
(2) *Actinobacillus pyogenes.*
(3) *Bacteroides melaninogenicus.*
(4) Fusobacterium *necrophorum.*
(5) Staphylococcus *intermedius.*

8. Examination of the left rear foot of a mature dairy cow reveals a red, papilloma-like lesion at the point of the heels. Which one of the following disorders is the most likely diagnosis?

(1) Pasture foot rot
(2) Hairy heel warts
(3) Laminitis
(4) Stable foot rot
(5) Benign foot rot

9. Laminitis in young dairy or beef cattle is considered to be associated with:

(1) *Fusobacterium necrophorum.*
(2) *Dichelobacter nodosus.*
(3) rumen acidosis.
(4) conformational defects.
(5) heredity.

10. Which statement best describes thrush in horses!

(1) An exudative dermatitis of the frog
(2) A yeast infection of the mouth
(3) A lymphangitis
(4) Laminitis
(5) Quarter cracks

11. Which one of the following statements regarding laminitis in horses is true?

(1) It seen most commonly in Draft breeds.
(2) The disorder is mainly a subclinical disease.
(3) Radiography best determines the diagnosis.
(4) Frequent walking of affected horses is the best treatment.
(5) It is a local manifestation in the foot of a systemic disturbance.

12. Which statement best describes infectious foot rot of sheep?

(1) It occurs most commonly in hot, dry environments.
(2) The disease is relatively simple to eradicate.
(3) It is the same as contagious ecthyma.
(4) This disease often responds to penicillin therapy.
(5) It causes lameness in carrier sheep.

13. Which one of the following statements regarding caprine arthritis-encephalitis (CAE) is true?

(1) The disease is not seen in North America.
(2) It demonstrates a high prevalence of seroconversion in affected herds.
(3) It is also known as mastitis, metritis, and agalactia syndrome.
(4) The disease produces a severe, sudden, debilitating lameness.
(5) Intra-articular antibiotics are the best treatment.

ANSWERS AND EXPLANATIONS

1. The answer is 5 [I A 1 5]. Creatine phosphokinase (CPK) is released from degenerating muscle cells and reaches very high (diagnostic) levels in the serum. Nutritional myopathies are caused by deficiencies of vitamin E and selenium. These myopathies occur in foals, lambs, calves, piglets, and mature ruminants. Disease is uncommon in the selenium-rich areas of the North American plains. Losses are most commonly caused by clinical disease.

2. The answer is 1 [I B 3 a]. Pressure necrosis and ischemic myopathy occur when large animals are recumbent on hard surfaces for extended periods. Milk fever and obturator paralysis may cause cows to become recumbent but are not the result of recumbency. Fat cow syndrome is a ketotic condition of obese cows, and blackleg is a clostridial myositis unrelated to recumbency.

3. The answer is 2 [I B 4]. These are different manifestations of the same inherited susceptibility for excessive skeletal muscle metabolism in swine. Blackleg and malignant edema are clostridial diseases of ruminants.

4. The answer is 3 [I B 4 g (2)]. Test matings have been replaced by a polymerase chain reaction gene probe and the other choices do not pertain to porcine stress syndrome.

5. The answer is 1 [I C 1 b (1)]. The most common presentation of blackleg in cattle is sudden death of growing beef animals on pasture. Wounds often are not present (as opposed to malignant edema), and immunization is preventive.

6. The answer is 5 [II A 3 b]. Although trauma may cause osteomyelitis, the most common cause is hematogenous spread from an infective process.

7. The answer is 4 [III A 1 c]. *Fusobacterium* necrophorum is a requirement for pasture foot rot (infectious pododermatitis) in cattle. Other organisms (e.g., *Bacteroides* melaninogenicus, Dichelobacter *nodosus*) may occasionally be recovered but are not necessary for disease. Actinobacillus pyogenes and Staphylococcus intermedius do not cause foot rot.

8. The answer is 2 [III A 4 b]. The clinical findings support a diagnosis of hairy heel warts (digital dermatitis).

9. The answer is 3 [III A 5 c (1)]. Interrupted feeding of high-grain diets causes bouts of rumen acidosis. This is thought to initiate the systemic hemodynamic changes that result in laminitis.

10. The answer is 1 [III B 11. Thrush is a disease of the foot of the horse. It is a moist dermatitis of the frog that emits a sour, foul odor. In advance cases, thrush may cause lameness.

11. The answer is 5 [III B 21. Diseases such as salmonellosis and endometritis may result in laminitis. Although laminitis may be irreversible in severe cases, early, vigorous therapy is often successful. Laminitis is diagnosed best by clinical findings, although radiology helps to provide prognostic information in more longstanding cases. Walking is controversial as a therapy but should not be considered in severely lame horses. Although often a subclinical disease in cattle, the condition is more often clinical in horses.

12. The answer is 4 [III C 6]. The causative organism, Dichelobacter nodosus, causes the most significant clinical signs in affected sheep. The organism is susceptible to penicillin therapy. The disease occurs most commonly on pasture under conditions of moisture. Although straightforward to treat, this disease is difficult to eradicate because the organism is protected in the feet of carrier sheep. These carrier sheep are not lame.

13. The answer is 2 [III D 1]. Caprine arthritis-encephalitis (CAE) is prevalent across North America, Europe, and Australia. It produces a chronic, progressive arthritis, and there is no known treatment.